# Pseudo-narcolepsy: case report

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This report describes the case of a 44-year-old woman presenting to a Sleep and Alertness clinic with symptoms of narcolepsy. The patient had clinical and polysomnographic features of narcolepsy, which disappeared after disclosure of severe psychological stress. Following a discussion of the differential diagnosis of narcolepsy, alternative diagnoses are considered. The authors suggest that the patient had a hysterical conversion disorder, or "pseudo-narcolepsy." Careful inquiry into psychological factors in unusual cases of narcolepsy may be warranted.

Ce rapport décrit le cas d'une femme âgée de 44 ans qui s'est présentée à une clinique de traitement des troubles du sommeil et de l'éveil avec des symptômes de narcolepsie. La patiente avait les caractéristiques cliniques et polysomnographiques de la narcolepsie, qui sont disparues après qu'on a découvert un stress psychologique grave. Après une discussion sur le diagnostic différentiel de narcolepsie, les auteurs envisagent d'autres diagnostics. Ils indiquent que la patiente avait un trouble de conversion hystérique ou une «pseudonarcolepsie». Une analyse attentive des facteurs psychologiques qui jouent dans des cas inusités de narcolepsie peut être justifiée.

## Case report

A 44-year-old married woman with a history of a recurrent depressive illness was referred to our Sleep and Alertness clinic by a neurologist. In the past, her depression had required antidepressant treatment and individual psychotherapy. Details of her family psychiatric or family medical history were difficult to obtain because she had been adopted at birth, but it was known that her biological mother had suffered from depression.

She gave an 18-month history of excessive daytime sleepiness (EDS), hypnogogic and hypnopompic hallucinations, cataplectic attacks and migraine headaches. Her cataplectic attacks consisted of a sudden bilateral loss of motor tone in her facial muscles and upper limbs, her head dropping forward and her eyes closing, and her not responding to her name being called. These attacks lasted minutes and were triggered suddenly by intense periods of emotion such as fear and anxiety. She did not have sleep paralysis or any automatic behaviours. In the initial consultation it was noted that "she likes to be ill." The neurologist was treating her headaches with an antimigraine medication. Computer-enhanced tomography and an electroencephalogram were normal.

An overnight polysomnographic sleep study with a Multiple Sleep Latency Test (MSLT) had been per-

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formed. The sleep study showed several polysomnographic features suggestive of narcolepsy: (1) a decreased total sleep time (5.8 hours); (2) 97 minutes of intervening wakefulness; (3) an impaired sleep efficiency (78%); and (4) rapid eye movement (REM) sleep within 30 seconds of sleep onset. In the MSLT, the patient had REM intrusions in naps 2, 3, and 4 (all within 2 minutes from lights out). She fell asleep in all 4 naps with a mean sleep latency of under 2 minutes. The presence of cataplexy and 2 or more REM onsets during the MSLT is considered diagnostic of narcolepsy.[1] Consequently, a diagnosis of narcolepsy was made, and the patient was treated with methylphenidate (20 mg twice a day) and clomipramine (100 mg at night).

Initially, treatment was effective and a Maintenance of Wakefulness Test showed a good improvement in the patient's alertness. However, after 6 months of treatment the patient began to experience side effects. Consequently, she stopped her medication. This resulted in a return of her sleep problems and coincided with a recurrence of her depressive illness.

She presented at the clinic, together with her husband, with hysterical symptoms and hypophonia. The staff interviewed the couple, and then had a discussion with her husband alone, during which admission was discussed. The patient was also then seen alone, at which point she disclosed that she had been having a sexual relationship with her general practitioner therapist (a hypnotherapist), which had placed her under severe stress for some time.

She was admitted to a psychiatric ward. On admission she had a labile mood, with underlying depression and poor concentration. Throughout the day she had regular cataplectic attacks, similar to the attacks she had had before admission. They now included sudden falls to the ground. Her migraine headaches had returned, and resulted in slurred speech and posturing of her left arm. These migrainous attacks lasted at least 20 minutes and often longer. The most likely diagnosis was still thought to be narcolepsy, but a focal epilepsy or migraine variant were also considered. An electroencephalogram showed normal results.

One week after admission, she disclosed the details of the affair with the hypnotherapist to the nurses on the inpatient unit. This disclosure to ward staff and her husband coincided with complete cessation of the cataplectic attacks and an improvement in her mood. (Her agreement to enter the inpatient unit was based on an understanding that her confidence would not be breached, but that she would disclose the information to the nurses as soon as she felt comfortable.)

A repeat sleep study, following the disclosure, showed a MSLT with no REM intrusions but mild hypersomnolence. She fell asleep in all 4 naps with sleep latencies of 9.0, 12.0, 8.0, and 7.0 minutes, giving a mean sleep latency of 9.0 minutes. The overnight polysomnogram showed a shortened REM latency consistent with depression. She was discharged without medication but received a course of cognitive behaviour therapy as an outpatient, concentrating on psychological issues secondary to relationships and her depressive illness. She continued to make good progress but still suffered from migraine headaches. Upon follow-up 2 years later there was no evidence of any narcoleptic features. The patient's mood is euthymic, and the migraine attacks have also abated.

## Discussion

This report summarizes the case of a 44-year-old woman presenting with symptoms of narcolepsy to the Sleep and Alertness clinic. After disclosure and subsequent treatment of severe psychological stress (sexual abuse by a treating physician), her symptoms disappeared.

The evidence supporting a diagnosis of narcolepsy included clinical symptoms (i.e., EDS, cataplexy, hypnogogic and hypnopompic hallucinations), overnight polysomnographic data, and an MSLT of less than 5 minutes with REM intrusion in 3 out of 4 naps.[1]

The more unusual features of this case, less supportive of a diagnosis of narcolepsy, were, first, the age of onset of symptoms. The onset of narcolepsy can often be traced back to a specific stressful life event.[2] However, this trigger usually coincides with an onset in the late teens or early 20s, and the narcolepsy is usually lifelong. Forty-four years of age is a relatively late age of onset, although onset has been reported to range from 12 years to 65 years of age, and a second smaller peak is observed in women nearing menopause.[3] Second, the diagnosis of cataplectic attacks was complicated by the patient's similar migraine episodes. Finally, the most conclusive evidence against the diagnosis of narcolepsy was the cessation of symptoms following the disclosure of her relationship with the hypnotherapist. As well, human leukocyte antigen (HLA) testing, performed with oligotyping, was negative for both DRw15 and DQw6. Only a very small percentage of patients with

narcolepsy test negative for these HLA types.[3]

Alternative diagnoses or explanations for the patient's sleep symptoms included hypersomnolence secondary to depression, hysterical conversion symptoms, false-positive or false-negative MSLT results, or side effects from the anti-migraine medication. Hypersomnolence, rather than insomnia, is reported to be present in some 20% of patients with depression.[4] However, most reports of hypersomnolence in depression have not been based on objective evidence. In a study that did report objective evidence, a group of patients with hypersomnia associated with mood disorders were compared with a group of patients with idiopathic hypersomnia. The MSLT and total sleep time results clearly distinguished the 2 groups; the patients with idiopathic hypersomnia had shorter MSLT values and longer total sleep times.[5] Furthermore, this patient's first overnight polysomnogram did not reveal the REM abnormalities that have been reported in patients with hypersomnolence secondary to depression.[3]

This patient may have had a hysterical conversion disorder or, as we have termed it, "pseudo-narcolepsy." Early attempts to understand narcolepsy considered EDS and cataplexy to be neurotic defences against emotional conflicts and viewed cataplexy as a conversion symptom.[6] Initially, the cataplexy may have been a "swoon" into her husband's arms; he describes an occasion, which they now date to be close to the onset of her infidelity, when she fell into his arms when he opened the door upon coming home. The secondary gain of repeating this behaviour to obtain his solicitous concern about her falling may have perpetuated the conversion process.

In an MSLT, 2 or more sleep-onset REM periods have been reported in normal subjects (i.e., those with sleep deprivation) and in patients with EDS but no cataplexy, i.e., without the full clinical picture of narcolepsy.[7,8] These patients may have a clinical variant of the classical presentation of the disease or have "evolving narcolepsy."[7] False-negative MSLTs can also occur. In 1 study, only 84% of patients with EDS and cataplexy had 2 or more sleep-onset REM periods during the MSLT.[9]

The anti-migraine medication the patient was taking (before the first sleep study only) contained phenobarbitone, ergotamine and atropine. Phenobarbitone has an overall sedating effect, decreasing sleep latency and wakefulness.[3] The effect of ergotamine on sleep architecture is unknown, but atropine and phenobarbitone are both known to suppress REM sleep.[10] The initial sleep findings, together with the response to treatment with methylphenidate and clomipramine are, therefore, not fully accounted for.

On first presentation, the patient was not clinically depressed, although her response to medication could have been due to the treatment of her hypersomnolence secondary to a masked depression. However, she had no polysomnographic evidence of depression on her initial overnight sleep study. It was initially assumed, when she was prescribed methylphenidate and clomipramine, that the improved alertness on a Maintenance of Wakefulness Test and the disappearance of the cataplectic attacks supported a diagnosis of narcolepsy. If this indeed was solely a conversion disorder, the response to medication remains unexplained.

Follow-up MSLTs, after the first negative MSLT, would have been useful, but the patient has, so far, remained free of clinical symptoms since she disclosed and dealt with the key severe psychological stressors. (The resolution of the situation included our mandatory reporting of the physician having a sexual relationship with the patient).

Hence, in unusual cases of narcolepsy, careful enquiry into psychological factors is perhaps more important than may have been previously appreciated.

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